[Primary Care]

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Common Leg Injuries of Long-Distance Runners: Anatomical and Biomechanical Approach

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Context: Long-distance running (greater than 3000 m) is often recommended to maintain a healthy lifestyle. Running injury rates increase significantly when weekly mileage extends beyond 40 miles cumulatively. With the development of running analysis and other diagnostic tests, injuries to the leg secondary to bone, musculotendinous, and vascular causes can be diagnosed and successfully managed.

Evidence Acquisition: Searches used the terms *running, injuries, lower extremity, leg, medial tibial stress syndrome, compartment syndrome, stress fractures, popliteal artery entrapment, gastrocnemius soleus tears*, and *Achilles tendinopathy.* Sources included Medline, Google Scholar, and Ovid from 1970 through January 2012.

Results: Tibial stress fractures and medial tibial stress syndrome can sometimes be prevented and/or treated by correcting biomechanical abnormalities. Exertional compartment syndrome and popliteal artery entrapment syndrome are caused by anatomic abnormalities and are difficult to treat without surgical correction.

Conclusion: Leg pain due to bone, musculotendinous, and vascular causes is common among long-distance runners. Knowledge of the underlying biomechanical and/or anatomic abnormality is necessary to successfully treat these conditions.

Keywords: running; jogging; leg injuries

Long-distance running (greater than 3000 m) is frequently recommended to maintain a healthy lifestyle.[121] Between 37% to 56% of recreational runners who steadily train and participate in a long-distance run periodically will sustain a running-related injury each year.[136] A retrospective study of adolescent runners (13-18 years) demonstrated a lifetime prevalence of previous running injury (68% in girls and 59% in boys) with strong associations with higher mileage and faster performance.[132] Known predictors of lower extremity injury include running greater than 40 miles per week and previous lower extremity injury.[52,56,141] While training errors lead to the majority of running injuries, biomechanical factors, such as foot insufficiency, muscle weakness, genu varum, and high Q-angle, contribute to 40% of running injuries.[79]

A recent systematic review of the incidence of leg injuries among long-distance runners found that most injuries involve the knee (7.2%-50%), lower leg (9%-32.2%), foot (5.7%-39.3%), or thigh (3.4%-38.1%).[135] The differential for leg pain is broad: skeletal (medial tibial stress syndrome, stress fractures), musculotendinous (tendinosis, myopathy), vascular (exertional compartment syndrome, venous thrombosis, popliteal artery entrapment syndrome, vascular claudication), neurologic (nerve entrapment, lumbosacral radiculopathy, neurogenic claudication), infectious, and neoplastic.[144] In a study of over 2000 running-related injuries, the most common leg injuries were medial tibial stress syndrome (4.9%), Achilles tendinopathy (4.8%), tibial stress fractures (3.3%), and gastrocnemius/soleus strains/tears (1.3%).[131]

Many of the skeletal, musculotendinous, and vascular running ailments can be explained by anatomy and basic biomechanics. A recent Cochrane review found little evidence for the effectiveness of stretching and/or conditioning for the prevention of lower limb soft tissue running injuries.[146] However, knee braces and custom insoles were effective for reducing anterior knee pain and medial tibial stress syndrome in runners, respectively.[146] Overall, evidence for interventions reducing lower limb pain and injury after intense running was considered weak.[146]

In addition to standard physical examination and imaging techniques (Table 1), new tools (running studies) have emerged

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Table 1. Common diagnostic testing findings.

Diagnosis: Modality	Findings
Chronic exertional compartment syndrome	
Compartment pressure testing	*Intracompartmental pressures:* > 15 mm Hg before exercise, > 30 mm Hg 1 min after exercise, > 20 mm Hg 5 min after exercise
Tibial stress fractures	
Magnetic resonance imaging	Cortical thickening, periosteal reaction, or fracture line
Bone scan	Focal uptake on all 3 phases
Medial tibial stress syndrome	
Bone scan	Diffuse linear uptake within the distal 1/3 of the posteromedial tibia on delayed images
Popliteal artery entrapment syndrome	
Duplex ultrasound	Decrease in peak systolic flow through popliteal artery with resisted plantar flexion and knee flexed to 15°
Magnetic resonance imaging/angiography	Medial deviation and compression, thrombosis, and/or aneurysm of the popliteal artery with an associated muscle anomaly (hypertrophied muscle, aberrant fibrous band, abnormal intercondylar origin of medial gastrocnemius)
Angiography	Medial deviation and precise, focal narrowing of the popliteal artery
Medial head of gastrocnemius strain/rupture	
Ultrasound	Partial or complete disruption of the medial gastrocnemius at the musculotendinous junction or fluid between aponeuroses of medial head of gastrocnemius and soleus without muscle rupture
Magnetic resonance imaging	Partial or complete disruption of fibers and hematoma at musculotendinous junction of medial head of gastrocnemius

for the detection and treatment of these injuries. Gait retraining is currently being studied as a viable option for preventing tibial stress fractures and other running-related injuries.[27,98]

BIOMECHANICS OF RUNNING

There are 3 phases of running gait—stance, swing, and float. As running speed increases, less time is spent in the stance phase.[40,77] Normal running gait begins with lateral heel strike, followed by foot pronation during midstance, and foot supination during push-off.[36,40] Proper running gait is critical to absorb the impact of striking the ground; insufficient or excessive pronation or supination alters force dissipation in the kinetic chain (muscles, ligaments, tendons, and bones).[40] In developed countries, most individuals wear running shoes and land on their heels.[77] The rear-foot strike running pattern is facilitated by elevated and cushioned heels of modern running shoes during which ground reaction forces reach 1.5 to 3 times body weight.[77] Three common recommendations include (1) motion control shoe for low arches, (2) cushioned trainer shoe for high arches, and (3) stability shoes for normal arches.[8,22] While these modifications have gained popularity within the running community to combat injury, there are currently limited data to suggest that these measures, based on arch type or foot shape, can reduce injury rates.[67,68,109]

Recently, biomechanics research has emerged to support the advantages of "barefoot" running. These runners generate smaller collision forces than individuals wearing standard cushioned running shoes.[77] The ability of "minimalist" runners to absorb impact more efficiently than shod runners may result from adaptations of dense plantar mechanoreceptors; the somatosensory feedback is likely diminished in shod runners.[111,112,125] To date, no clinical studies substantiate the claims of injury reduction using a "minimalist" style.

VIDEO GAIT ANALYSIS

While running gait is highly variable, there are certain abnormalities correlated with injury.[36] Dynamic gait analyses allow for identification of biomechanical factors not obvious

with standard static examinations.[51] More sophisticated methods, such as electromyography, accelerometers, electrogoniometers, and gyroscopes, are generally reserved for research purposes.[51]

Observational gait analysis (ie, patient walking in the hallway) can provide some useful information but is fraught with error because multiple motion segments are difficult to assess simultaneously.[105] Viewing in multiple planes provides the truest picture of running form.[105] Videotape observational gait analysis (VOGA) enhances accuracy of gait analyses and is performed from head to toe using anterior, posterior, and lateral views on a standard treadmill.[99] Force plates determine precise kinetic and kinematic forces. Treadmill analyses are applicable to ground running[110] within 1 standard deviation, if the treadmill surface is sufficiently stiff and belt speed regulated.[89] While gait analysis may help identify biomechanical abnormalities, it is unclear if these corrections will prevent or help treat lower extremity injuries.

MEDIAL TIBIAL STRESS SYNDROME

Medial Tibial Stress Syndrome, Anatomy and Biomechanics

Medial tibial stress syndrome is thought to be a periostitis caused by abnormal traction by the deep flexor[43] and/or soleus muscles.[32] A recent review cautions that histologic studies have failed to provide evidence for periostitis as an underlying etiology. Mismatch between bony resorption and formation with resultant overloading of the tibial cortex is a likely etiology of medial tibial stress syndrome.[94] Anatomic evidence suggests that either the soleus or an aponeurotic band connects the medial soleus to the posteromedial tibia and can impart traction stress to the periosteum when the soleus contracts and stretches.[15,58,86]

Static and dynamic measurements have been studied to determine which anatomical (limb length, ankle dorsiflexion, first metatarsophalangeal joint extension, and arch height) and biomechanical (center of pressure excursion, malleolar valgus index, and gait velocity) factors were associated with medial tibial stress syndrome.[12] Subjects with medial tibial stress syndrome had significantly greater visual analog pain levels and slower gait velocity.[12] Medial tibial stress syndrome is associated with an imbalance of foot pressure (greater on the medial foot), excessive pronation, sudden increase in intensity and/or duration of training, and an uneven training terrain.[103,120] These factors increase soleus strain by eccentric contraction to resist pronation. In a separate study, decreased hip internal rotation, increased ankle plantar flexion, and positive navicular drop were associated with medial tibial stress syndrome.[92]

Nonoperative Treatment

Treatment strategies for medial tibial stress syndrome frequently include rest and cross-training using low-impact activities such as stationary biking and underwater running.[38] Once symptoms resolve, training should slowly accelerate (10% to 25% every 3 to 6 weeks).[73]

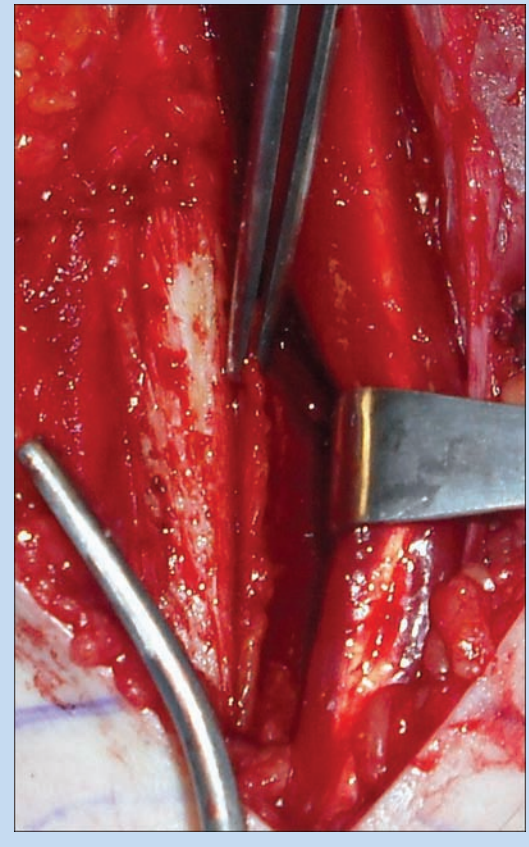

Figure 1. Posteromedial tibial stress syndrome is treated by release of the deep posterior leg fascia and excision of the posteromedial tibial periosteum (held by forceps).

The efficacy of physical therapy and nonsteroidal anti-inflammatory drugs is questionable. In military recruits, aspirin, phenylbutazone, heel-cord stretching exercises, and/or short walking cast for 1 week provided no significant decrease in the duration of shin splints compared to rest alone.[6] Off-the-shelf orthotics and calf stretching can improve medial tibial stress syndrome, even with chronic pain.[78] Extracorporal shockwave therapy has shown promise; the value of the pneumatic leg brace is unknown.[91,93] Circumferential straps[19] and taping[55] have not been shown to effectively dampen posteromedial tibial stress. Unfortunately, current evidence does not support any prevention method for medial tibial syndrome.[26]

Operative Treatment

Surgical outcomes studies on medial tibial stress syndrome are of poor methodological quality and design.[94,137] The most effective surgical procedures involve release of the deep posterior compartment, including the soleus sling and removal of a strip of posteromedial tibia periosteum (Figure 1).[32,145] A recent series of operatively treated cases noted good/excellent results in 69% and fair/poor results in 31% at 30 months

postoperatively.[145] Only 41% returned to their previous level of athletic activity.[145] Complications occur frequently (hematoma, localized paresthesias, numbness, and stress fractures).[32,60,145]

NONINSERTIONAL ACHILLES TENDINOPATHY

Anatomy and Biomechanics

During running, the Achilles tendon loading exceeds 12.5 times the runner's body weight.[69,70] The gastrocnemius-soleus complex allows the Achilles tendon, the strongest tendon in the body,[59,62] to absorb tremendous energy before strain injury occurs.[59,62] The tendon fibers spiral 90° instead of vertically increasing potential elongation and energy production.[2,97] The tendon can stretch up to 4% before microscopic damage occurs; macroscopic rupture occurs at strain levels greater than 8%.[61]

Factors influencing Achilles tendinopathy are both intrinsic and mechanical. Intrinsic factors (regional hypovascularity, endocrine or metabolic diseases, genetic makeup) can predispose the tendon to degeneration.[137] Mechanical factors (overuse and lack of flexibility) increase tendon strain surpassing the energy-absorbing ability and leading to microtears.[137] Training errors (technique, inappropriate footwear, inconsistent surfaces) are associated with degeneration.[62,137] Excessive lateral heel strike and compensatory foot pronation can result in uneven force generation from the gastrocnemius and soleus muscles and overloading certain areas of the Achilles tendon.[57,124] Altered knee kinematics and reduced proximal muscle activity (rectus femoris and gluteus medius) are associated with Achilles tendinopathy.[9,35]

Nonoperative Treatment

Relative rest and nonsteroidal anti-inflammatory drugs reduce pain produced by inflammation in the acute phase; physical therapy and orthoses can correct underlying mechanics contributing to this injury.[24,25,83,101,102] Total abstention from running is recommended acutely; later in the disease process, light loading may be helpful in stimulating healing and remodeling of collagen fibers.[80,142] Evidence for deep friction massage, soft tissue mobilization, and stretching is limited[28,45]; therapeutic techniques should incorporate eccentric strengthening of the triceps surae.[3,4,64,127]

A recent review of conservative treatments for chronic Achilles tendinopathy found strong evidence for heavy load eccentric exercise.[118] Nonsteroidal anti-inflammatory drugs and shock wave therapy are not proven treatments, corticosteroid injections should be avoided,[118] and other injections (ie, platelet-rich plasma injections) are still experimental.[118] The majority of patients with Achilles tendinopathy recover fully with exercise alone.[123] Reduced neuromuscular activity of the gastrocnemius muscle during weightbearing in patients suffering with Achilles tendinopathy should be a focus of treatment.[13] Heel lifts of 12 to 15 mm can decrease strain in the Achilles tendon pain[25,95] and resolve symptoms in up to 75% of runners.[119,122,139] Devices that limit hindfoot eversion may also avert Achilles tendinopathy by preventing excessive pronation during midstance[115]; braces and splints have not been beneficial.[118]

Operative Treatment

Several techniques are available for Achilles tendinopathy and share basic tenets: longitudinal incision within the tendon to detect lesions and excision of fibrotic adhesions and degenerated nodules.[80] If large lesions are excised, reconstructive procedures, including local transfers (flexor hallucis longus), may be necessary.[143] Surgical success rates of 70% to 85% have been reported.[130] In the largest follow-up study to date, Paavola et al noted that 11% of patients had postoperative complications with 3.2% requiring additional procedures. The most common complications were skin necrosis and wound infection.[102]

To reduce the risk of skin-related problems, outpatient percutaneous longitudinal tenotomy has been introduced.[81] This procedure decreases tendon strain and improves local circulation for better nourishment and healing.[41,81,133] In middle- and long-distance runners, 37 of 48 patients reported excellent[25] or good[12] results at an average of nearly 2 years following this procedure.[81]

TIBIAL AND FIBULAR STRESS FRACTURES

Anatomy and Biomechanics

The tibial shaft is the most common site of lower extremity stress fracture in runners and accounts for nearly 50% of all stress fractures in athletes.[48] Stress fractures along the anterior cortex are associated with the radiographic "dreaded black line" and sluggish healing often necessitating operative treatment (Figure 2). Fibular stress fractures occur less frequently and represent 4.6% to 21% of all athletic stress fractures.[47,96]

Normally, bone remodels to match demands according to Wolf's law.[48] When under repetitive loads without sufficient time to remodel, bone will fatigue and fail.[48] The initial microdamage inevitably propagates into macroscopic damage if stress continues (Figure 3).[63] In the tibia, the resulting stress fracture usually occurs on the compression side and involves the posteromedial cortex in a transverse orientation.[17] High-risk tibial stress fractures occur less frequently along the anterior cortex of the midtibia or medial malleolus. The mechanism of injury may be repetitive ankle plantar flexor contractions.[48] Fibular stress fractures typically occur within the shaft, 5 to 6 cm proximal to the lateral malleolus corresponding to the attachment site of these same tendons.[21]

Risk factors for tibial stress fractures in male runners are difficult to predict; low bone mineral density and lean mass in lower limb, menstrual imbalance, and low-fat diet are associated with stress fractures in female runners.[16] Menstrual irregularities and high weekly training mileage place runners at high risk of recurrence.[72] Biomechanical abnormalities (leg-length discrepancy, peak hip adduction, rearfoot eversion

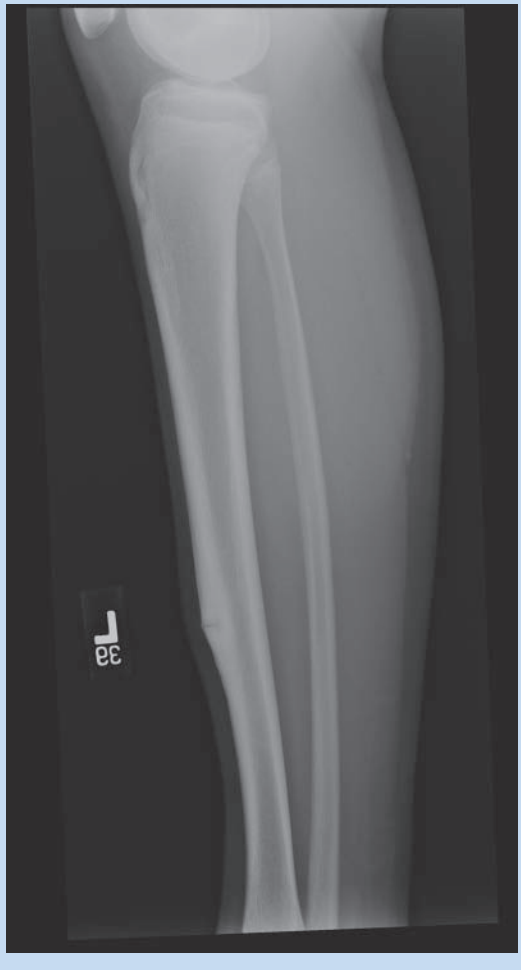

Figure 2. Lateral radiograph of the tibia demonstrates "dreaded black line," which is a poor prognostic indicator of spontaneous healing.

angles during stance phase of running) have also been linked to tibial stress fractures in women.[39,106] Vertical instantaneous load rate and peak knee adduction and internal rotation forces do not appear to play a role in development of tibial stress fractures.[106]

Systemic reviews have failed to definitively link foot type with tibial stress injury. However, extremes of foot type, including high longitudinal arch and excessive forefoot varus, may predispose runners to tibial stress fractures.[11] Running on a treadmill and longer foot pronation lessen the risk of developing a tibial stress fracture.[50,88]

Nonoperative Treatment

Fibular and low-risk tibial stress fractures often respond to a period of rest ranging from 4 to 8 weeks.[48,63] Low-risk stress fractures occur in locations that have adequate vascularity and are under less strain than high-risk stress fractures (eg, anterior tibia, tension-side of femoral neck, navicular, base of the fifth metatarsal). Progressive return to sports is initiated with cross-training followed by higher impact activities.[41,54]

A variety of interventions have been proposed to hasten return to play and/or assist in the treatment of tibial stress fractures with a propensity for sluggish healing.[48] Coupled electric fields and extracorporeal shock wave therapy may improve treatment of these fractures[14,96]; pneumatic leg braces show conflicting results.[5,129] Pulsed ultrasound[114] and bone stimulators[147] have not been effective. Bisphosphonates may accelerate the healing process for tibial stress fractures[128] and/or preventing their occurrence.[87]

Operative Treatment

Despite appropriate treatment, some tibial stress fractures fail to heal and result in the "dreaded black line," sclerosis, and/or cyst formation.[147] The anterior, tension-sided tibial stress fractures are at an elevated risk for delayed healing (presumably from hypovascularity). However, there is still a role for conservative care: operative intervention does not guarantee healing and may lead to complications such as infection, pain at the nail insertion site, or completion of the fracture.[147]

The surgical treatment of choice is an intramedullary nail.[23] Alternately, drilling with bone grafting has shown favorable results.[90] These techniques may allow for faster return to sports with minimal morbidity.[90]

GASTROCNEMIUS-SOLEUS STRAIN/ RUPTURE

Anatomy and Biomechanics

The multipennate gastrocnemius and soleus muscles are commonly strained due to their complex structure,[20] accounting for 3.6% of all reported soccer injuries.[7] The medial head of the gastrocnemius originates from the medial femoral condyle and fuses with the smaller lateral head before joining the soleus aponeurosis to form the Achilles tendon.[20] The gastroc is a "fast action" muscle composed largely of type IIb fibers and spans 2 joints, making it susceptible to strain injuries.[31] The soleus only crosses the ankle joint and consists largely of type I slow-twitch fibers and is less likely to be injured.[20]

Injury to medial head of the gastrocnemius (Figure 4) is caused by sudden dorsiflexion of a plantar flexed foot with the knee in extension or sudden extension of the knee with the ankle dorsiflexed.[89] Running studies indicate that this injury occurs near touchdown and is associated with faster-than-normal running speeds and inappropriate body posture, which causes altered muscle length and shock absorption.[71] The injury has a predilection for the poorly conditioned, middle-aged athlete with "thick calves" who is engaged in strenuous activity.[74]

Nonoperative Treatment

Medial head of gastrocnemius and soleus strains are treated nonoperatively with a combination of ice, analgesics, and

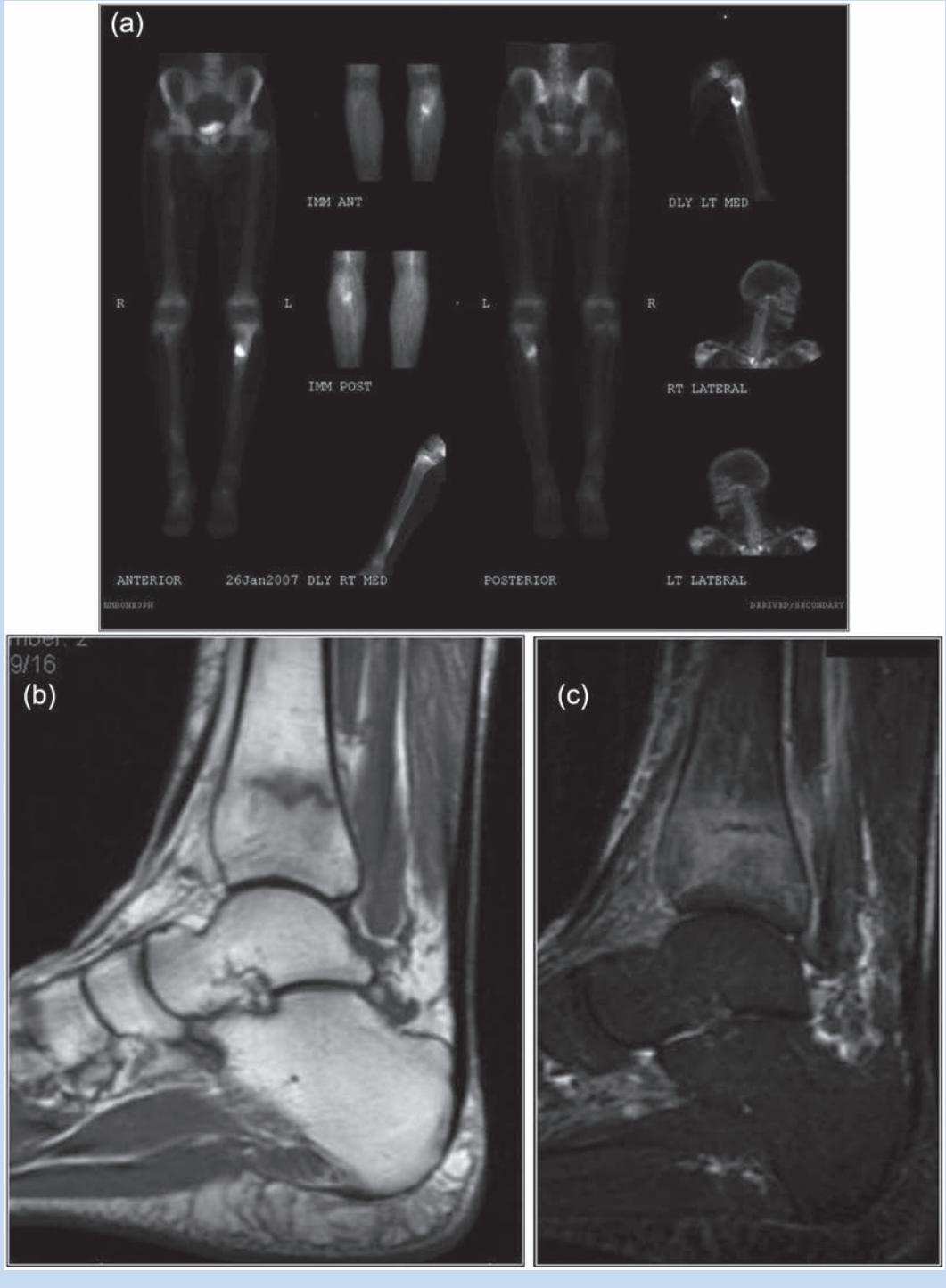

Figure 3. (a) Bone scan demonstrates increased tracer uptake in the proximal tibia in both immediate and delayed phase images consistent with a proximal tibial stress fracture. (b) and (c) Magnetic resonance imaging clearly shows a distal tibial stress fracture line on both T1 and T2 weighted images.

casting/splinting (night splint or CAM boot), depending on the extent of injury. In severe strains and ruptures, casting in plantar flexion for 3 weeks, followed by neutral boot/cast for an additional 3 weeks, may suffice.[89] Dorsiflexion to neutral is expected at 1-month postinjury, while return to preinjury level may be achieved by 2 months.[84,85] Ultrasound can be used to confirm healing.[74] Compression of the limb decreases hematoma size and facilitates healing.[75]

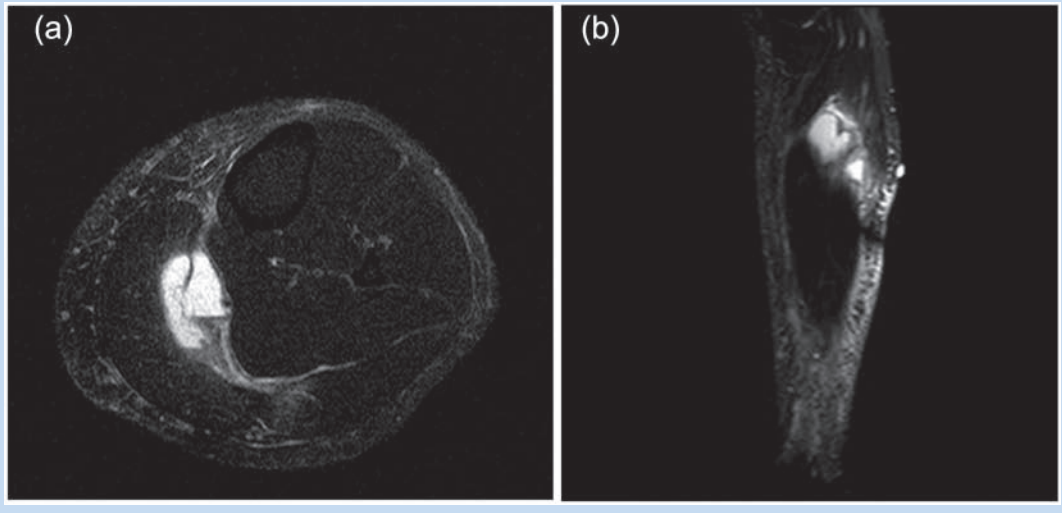

Figure 4. (a) and (b) Axial and sagittal T2-weighted magnetic resonance images demonstrate a fluid collection at the musculotendinous junction of the medial head of the gastrocnemius correlating with a tear.

Operative Treatment

Although controversial, surgical repair for complete ruptures of the medial head of the gastrocnemius may be indicated acutely in those unable to maintain full body weight on the metatarsal heads with the ankle in maximal plantar flexion and/or a palpable defect.[89] The surgical technique involves direct reapproximation of the tendon edges at the musculotendinous junction.[89] Fasciotomy is indicated if a large hematoma precipitates a compartment syndrome.[31,74]

CHRONIC EXERTIONAL COMPARTMENT SYNDROME

Anatomy and Biomechanics

During heavy exercise, fluid accumulates within the interstitial space of skeletal muscle,[107] increasing mass up to 20%.[116] The buildup of interstitial fluid combined with limited expansion of the fascial compartments, especially the anterior and lateral leg compartments, may lead to elevated intramuscular pressures, causing capillary occlusion.[107] The decreased blood flow can result in cell hypoxia, increased dependence on anaerobic metabolism, production of lactate,[107] and eventual cell death. Recent studies have failed to link stiffness and thickness of muscle fascia to chronic exertional compartment syndrome.[29] However, low muscle capillary supply is a possible pathogenic factor.[37]

There are limited data on risk factors for chronic exertional compartment syndrome. The mechanism remains elusive though landing style,[65] muscle type composition,[140] and capillary density within skeletal muscle[37] may be contributors.

Nonoperative Treatment

Cessation of the inciting activity remains the only nonoperative measure to effectively treat chronic exertional compartment syndrome long-term.[42] In cases when elevated postexercise compartment pressures are associated with abnormally increased distal vascular resistance (decreased postexercise ankle-brachial index, biphasic arterial waveforms in distal extremity, slow-flow velocity), lumbar sympathetic blockade has been proposed as a method of increasing blood flow to muscle during exercise.[44] With a lumbar sympathetic block, the normal reflex sympathetic adrenergic-mediated vasoconstrictive effect in vessels supplying the working muscle is blunted, so the vasodilating effects of accumulating local metabolites predominates.[44]

Operative Treatment

Fasciotomy is the mainstay of treatment for those athletes who wish to return to sporting activities.[32,42,100] The procedure involves releasing the fascia overlying the anterior and lateral compartments and, if necessary, the superficial and deep posterior compartments. While a variety of fasciotomy techniques have been described, 78% to 92% of athletes can expect to return to their previous level of function.[42,53,138]

Less invasive techniques such as endoscopic and percutaneous fasciotomies using a fasciotome have been developed to minimize surgical incision size and soft tissue disruption and allow swifter return to sports.[42,66] Despite these advantages, minimally invasive techniques may inadequately release and/or increase risk of injury to neurovascular structures (superficial peroneal nerve, saphenous nerve, and saphenous vein).[54]

A structured rehabilitation program maximizes functional outcome after surgery.[117] Patients are progressed from "PRICE" to full range of motion by focusing on soft tissue mobility via stretching, neurodynamic mobilization, strengthening, and ultimately biomechanical analysis of the athlete during sport specific activities.[117]

POPLITEAL ARTERY ENTRAPMENT SYNDROME

Anatomy and Biomechanics

Before bifurcating into the anterior and posterior tibial arteries, the popliteal artery normally courses beneath and between the medial and lateral heads of the gastrocnemius, adjacent to the plantaris and popliteus muscles, and through the tendinous arch of the soleus.[10] Alterations of the normal structural relationships can cause compression of the popliteal artery.[47,108] Most cases of popliteal artery compression are caused by anatomic variations formed during embryonic stages of development[30] and include an abnormal attachment of the medial head of the gastrocnemius within the intracondylar notch,[104] medial course of the popliteal artery,[1] and/or aberrant fibrous bands.[1,113] More recently, a "functional" variation has been attributed to hypertrophy of surrounding musculature.[10] Care must be taken to distinguish popliteal artery entrapment syndrome from chronic exertional compartment syndrome, as the 2 disorders occur in the same population and present with similar symptoms.[134]

In the development of popliteal artery entrapment syndrome, the popliteal artery is focally compressed against the medial femoral condyle during forceful plantar flexion.[27,40] With repeated constriction, the artery can sustain damage to its wall and form aneurysms and/or stenotic lesions leading to thrombosis and/or embolic events.[27,40]

For reasons unknown, men are more prone to the disease.[47,126]

Nonoperative Treatment

Because of the progressive nature of the entrapment, nonoperative management has a limited role. Early detection and intervention can limit progression of the process and lead to more favorable outcomes by minimizing arterial damage.[34,82,148]

Operative Treatment

While thrombolytic therapy is controversial in limiting the extent of subsequent bypass grafting,[148] surgical intervention is the hallmark of treatment for ischemic symptomatic patients.[18,100] Decompression of the lesion by division of the medial head of the gastrocnemius is often necessary to prevent recurrence.[34,82,148]

The need for vascular surgery depends on the chronicity of the vascular compression.[76,148] In long-standing entrapment, the popliteal artery may sustain irreversible vessel wall damage and is prone to atherosclerosis, aneurysm, and thrombosis.[49] Bypass grafting appears to be the treatment of choice.[46,47,76] Vein patching and endovascular treatments have had limited success.[49]

Many patients return to previous activities following surgical intervention[113]; those with decompression alone tend to have better outcomes than decompression plus reconstruction.[34,82]

CONCLUSION

Chronic exertional compartment syndrome, tibial stress fractures, posteromedial tibial stress syndrome, popliteal artery entrapment syndrome, gastrocnemius-soleus strains/tears, and Achilles tendinopathy are common running ailments. While no clear evidence exists that these injuries can be prevented, most can be treated successfully by considering underlying anatomical and biomechanical causes. The majority of tibial stress fractures, gastrocnemius-soleus strains/tears, and Achilles tendinopathy are effectively managed with an appropriate balance of relative rest and therapy. Chronic exertional compartment syndrome and popliteal artery entrapment syndrome, both thought to be caused by anatomic abnormalities, are often remedied by surgical intervention.

Clinical Recommendations

SORT: Strength of Recommendation Taxonomy
A: consistent, good-quality patient-oriented evidence
B: inconsistent or limited-quality patient-oriented evidence
C: consensus, disease-oriented evidence, usual practice, expert opinion, or case series

Clinical Recommendation	SORT Evidence Rating
There is little evidence for the effectiveness of stretching and/or conditioning for the prevention of lower limb soft tissue running injuries.[146]	B
Heavy load eccentric exercise is recommended for chronic Achilles tendinopathy.[3,4,64,118,127]	B
Fibular and low-risk tibial stress fractures (posteromedial tibial cortex) often respond to a period of rest, ranging from 4 to 8 weeks.[48,63]	C
Operative interventions are recommended for chronic exertional compartment syndrome (fasciotomy) and popliteal artery entrapment syndrome (division of the medial head of the gastrocnemius).[31,33,41,81,147]	C

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